1062-73 Evaluation of Coronary Flow Characteristics in Patients with Hypertrophic Cardiomyopathy Using A Doppler guidewire

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Hemodynamic parameters of coronary flow, including its pattern and flow reserve, were evaluated in 19 patients with hypertrophic cardiomyopathy (HCM) using a doppler guide wire (Cariometrics, inc.) Results were compared with those obtained in 20 control group. The average peak velocity (APV) of RCA and LAD in HCM were 28.2 ± 8.2 and 31.8 ± 7.2 cm/sec respectively. These values were higher than those of the control group. The diastolic systolic velocity ratio (DSVR) of LAD in HCM was significantly greater than that of the control group (10.2 ± 3.2 vs 3.6 ± 0.8, $p < 0.01$). Coronary flow reserve in HCM was slightly lower than that of the control group (2.4 ± 1.0 vs 2.7 ± 0.5, ns). The early % one third time velocity integral (% 1/3) was significantly decreased in the HCM group (38 ± 11 vs 49 ± 14%, $p < 0.05$). The coronary flow volume calculated by vascular diameter and coronary flow velocity was significantly increased in HCM (194 ± 100 vs 103 ± 41 ml/min, $p < 0.01$), however the flow volume/left ventricular mass ratio was lower in HCM compared with that of the control group (0.64 ± 0.21 vs 0.73 ± 0.31). The coronary flow of patients with HCM was characterized by a higher velocity and greater flow volume; however, the flow per left ventricular mass was decreased.

1062-74 The Coronary Vasodilator Reserve is More Severely Impaired in Patients With Hypertrophic Cardiomyopathy and Left Ventricular Dysfunction

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Some patients (pts) with hypertrophic cardiomyopathy (HCM) develop progressive dilatation of the left ventricle (LV) with systolic dysfunction. In HCM, the coronary vasodilator reserve (CVR) is impaired despite angiographically normal coronaries. Aim of this study was to ascertain whether a correlation exists between the severity of CVR impairment and the development of LV dysfunction in HCM. *Methods:* Eighty-four HCM pts (62 males, aged 42 ± 12 years) were studied. LV end-diastolic (LVEDD) and end-systolic diameters (LVESD) were measured by 2D-echo, and fractional shortening (FS) was computed as (LVEDD-LVESD)/LVEDD × 100. Myocardial blood flow (MBF; ml/min/g) was measured at baseline (bas) and after dipyridamole (dip; 0.56 mg/Kg) with positron emission tomography and N13-ammonia or O15-water. CVR was then computed as dipMBF/basMBF (absolute units). *Results:* In our series, 53 pts were in NYHA class I, 24 in class II and 7 in class III; 12 patients had an abnormal FS. Flow data are shown in the table.

	NYHA I	NYHA II	NYHA III
bas MBF	0.83 ± 0.27	0.91 ± 0.37	0.70 ± 0.22
dip MBF	1.57 ± 0.64	1.52 ± 0.58	0.96 ± 0.32
CVR	1.93 ± 0.64	1.69 ± 0.54	1.40 ± 0.43

Pts in advanced NYHA classes had lower dip MBF and CVR (ANOVA, $p < 0.05$). A negative correlation was found between dip MBF and LVESD ($R = 0.23$; $p < 0.05$), and dip MBF was lower in pts with abnormal FS (1.54 ± 0.65 vs 1.86 ± 0.60; $p < 0.05$). *Conclusions:* In pts with HCM, a relationship between the severity of microvascular derangement and evolution to LV dysfunction can be demonstrated.

1062-75 Hypertrophic Cardiomyopathy as an Important Cause of Sudden Cardiac Death on the Athletic Field in African-American Athletes

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Sudden death in young competitive athletes is due to a variety of congenital heart malformations, most commonly hypertrophic cardiomyopathy (HCM). Because athletic field deaths are an important issue in the African-American community, we studied the relation of race to the causative cardiovascular lesion in 153 competitive athletes who died suddenly, aged 9–40 years (mean 17); 84 athletes (55%) were white, 64 (42%) were black and 5 (3%) Asian or Native American. Of these 153 athletes, most (49; 32%) died of HCM or anomalous coronary artery (20; 13%). HCM was a significantly more common cause of sudden death in black athletes (28/64; 44%) than in white athletes (21/84; 25%; $p = 0.02$). Of the 28 black athletes with sudden death, most (24; 84%) participated in basketball or football; all were male. No statistically significant differences in occurrence were identified between the races for other diseases. A separate analysis of the standard international HCM-cardiology literature, 1970–94, identified 2174 HCM patients in 46 papers, and none were reported to be black. In conclusion, 1) HCM appears to be a significantly more common cause of sudden death on the athletic field in young black athletes compared to white athletes; 2) the substantial occurrence of HCM-related sudden death in young black male athletes contrasts sharply with the infrequent identification of HCM in hospital-based populations; 3) in black males, HCM is most frequently encountered when it is a cause of sudden and unexpected death on the athletic field.

1062-76 Increased Plasma Levels of Adrenomedullin in Patients with Hypertrophic Cardiomyopathy

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Adrenomedullin (AM) is a novel peptide recently isolated from pheochromocytoma, eliciting vasorelaxant activity. The aim of this study is to elucidate whether AM plays an important role to maintain hemodynamics in patients with hypertrophic cardiomyopathy (HCM). The radioimmunoassay for AM was performed with adrenomedullin-M antibody. Plasma level of AM was measured in 40 patients with HCM [14 hypertrophic obstructive cardiomyopathy (HOCM), 57 ± 14 yrs, 26 hypertrophic nonobstructive cardiomyopathy (HNCM), 58 ± 13 yrs] and 14 normal control subjects (NC: 53 ± 15 yrs). Plasma norepinephrine (NE), atrial and brain natriuretic peptides (ANP & BNP) were also measured. Total 12-lead QRS voltage was measured as a marker of left ventricular hypertrophy.

	NC	HOCM	HNCM
AM (pmol/L)	5.2 ± 0.4	9.7 ± 5.6**	7.8 ± 1.9*
NE (ng/L)	319.9 ± 71.7	297.4 ± 189.3	337.6 ± 153.4
ANP (ng/L)	11 ± 6	99 ± 75**#	40 ± 28*
BNP (ng/L)	6 ± 3	431 ± 296**#	212 ± 300*

*$p < 0.05$ vs NC, **$p < 0.001$ vs NC, #$p < 0.0002$ vs HNCM.

Plasma levels of AM were significantly higher in HCM (8.43 ± 3.73 pmol/L, $p < 0.003$) than in NC (5.24 ± 0.44). There was a significant correlation between plasma level of AM and total 12-lead QRS voltage in HCM ($r = 0.323$, $p < 0.05$). There were no significant correlations between plasma AM and NE, ANP or BNP.

In conclusion, AM may play an important role to maintain hemodynamics in patients with HCM, independent of NE, ANP and BNP.

1062-77 Left ventricular outflow tract gradient a potential cause for dyspnea in the elderly

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Limitation of exercise tolerance by breathlessness is common in the elderly, although LV cavity size and systolic function are normal. To assess the role of dobutamine stress echo, we studied 30 consecutive pts with exertional dyspnea and negative exercise stress test, age 70 ± 12 yrs, 21 female, with M-mode, 2-D and Doppler-echo. Resting results were compared with 12 normals, age 69 ± 10 yrs. *Before stress:* LV EDD and ESD were normal but basal septum 2.3 ± 0.5 vs 1.3 ± 0.2 cm and posterior wall 1.2 ± 0.2 vs 0.9 ± 0.1 cm were thickened, $p < 0.001$ (vs normal). Peak LV thinning rate was reduced 8.1 ± 3.5 vs 10.4 ± 2.6 cm/s, $p < 0.05$, and transmitral A wave velocity increased 0.86 ± 0.1 vs 0.62 ± 0.1 m/s, $p < 0.001$. *At peak stress:* heart rate rose from 80 ± 12 to 132 ± 26 beat/min and systolic blood pressure from 135 ± 15 to 170 ± 14 mmHg, $p < 0.001$, but LV cavity dimensions did not change. Peak LV outflow tract velocity increased 4.2 ± 1.2 m/s vs 1.5 ± 0.5 m/s (at rest), systolic mitral leaflet septal distance reduced from 13 ± 4.5 to 2.2 ± 1.9 mm, $p < 0.001$, with SAM in 24 (80%) pts. Transmitral E wave velocity did not change though A wave velocity increased from 0.84 ± 0.24 to 1.1 ± 0.2 m/s, $p < 0.001$. All pts developed dyspnea at peak stress, but none a new wall motion abnormality.

Conclusion: The combination of exercise limiting breathlessness and normal LV systolic function is common in the elderly. Ventricular filling maintained with stress excludes diastolic heart failure. However, the close correlation with symptoms suggests that high outflow tract velocities associated with basal septal hypertrophy is the underlying mechanism.